

THE RELATIONSHIP BETWEEN PAIN AND MOOD:
DOES MOOD PREDICT REPORTS OF ACHES AND PAINS IN A HEALTHY SAMPLE?

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Abstract

The current study examined the predictive relationship between positive affect (PA), negative affect (NA) and reports of general aches and pains. Because little is known about the relationship between emotion arousal levels (e.g. high-arousal and low-arousal) and pain, secondary analyses were done investigating whether emotional arousal, especially within the PA domain, predicts lower pain reports. Eighty-three healthy undergraduates (mean age = 18.29; 44.6% men, 55.4% women; 66.3% Caucasian) participated in a 13-day longitudinal study using an ecological momentary assessment (EMA) design to evaluate participants' mood and pain reports four times a day, each of the 13 days. Mood was evaluated using an adjective checklist of emotion words from both the Profile of Mood States (POMS) and the Positive and Negative Affect Schedule (PANAS), and pain was measured using the Cohen-Hoberman Inventory of Physical Symptoms-Short Form (CHIPSS-SF). Within the 2-level models, only daily PA was predictive of next-day pain, with increased PA predictive of less pain. However, trait neuroticism drove the other models in predicting higher levels of next-day pain when neuroticism was high. In the 3-level models, PA, NA, and both high- and low-arousal PA were predictive of within-day pain in the directions expected. Neuroticism was a powerful predictor of within-day pain as well. Implications from this study include the importance of multiple components of mood on the pain experience in the moment, but only general PA seems to matter over a longer period of time (days). More research should be done investigating the particular role of high- and low-arousal PA in symptom reports and the pain experience in order to identify mechanisms of action and when these mechanisms are most potent.

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The Relationship between Pain and Mood:

Does Mood Predict Reports of Aches and Pains in a Healthy Sample?

Much attention has been paid to the role of negative affect (NA) and pain with over 50 studies examining the association in various capacities (e.g. Berkowitz & Thome, 1987; Feeney, 2004; Frew & Drummond, 2007; Gaskin, Greene, Robinson, & Geisser, 1992; Gedney & Logan, 2007; Janssen, 2002; Kratz, Davis, & Zautra, 2007; Lightsey et al., 2009; O'Brien et al., 2008; Pauli, Wiedemann, & Nickola, 1999; Rhudy & Meagher, 2003; Stalling, 1992; Staud, Price, Robinson, & Vierck, 2004). For example, a number of studies have investigated the role of depression and anxiety on pain, with findings that individuals with depression and anxiety disorders had more frequent, intense, and unpleasant pain reports than healthy controls or participants without chronic pain conditions (Bair, Robinson, Katon, & Kroenke, 2003; Dworkin & Gitlin, 1991; Dworkin, Von Korff, & LeResche, 1990; Lautenbacher, Sernal, Schreiber, & Krieg, 1999; Schieir et al., 2009; Tait, Chibnall, & Margolis, 1990; Vaccarino, Sills, Evans, & Kalali, 2009).

Despite the research attention focusing on NA and pain, little consideration has been paid to the role of positive affect (PA) in the pain experience. There are two plausible explanations for this absence. First, the issue of pain has been predominated by the medical model, or specifically, a focus on disorder and disease and what is *wrong* with people, as opposed to studying what is *right* (i.e., positive). Because researchers and clinicians alike focus on factors influencing these negative experiences, the role of PA is often forgotten and left unmeasured in studies of pain as well as other health outcomes (Pressman & Cohen, 2005).

The second possible reason for the relative absence of PA in pain literature is due to a lack of consensus in the definition of both PA and the structure of emotion. Because neither PA

nor emotion have agreed-upon definitions in the literature, it is difficult to determine a clear understanding of the relative relationships between NA, PA, and pain.

Emotion

Because of the sheer complexity and depth of the emotion literature, it is first important to understand some of the terms that will be addressed in this paper. Emotion, mood, and affect may all be used interchangeably. When describing the emotion literature, emotion is used as a description of a transient feeling at that moment, while mood, as well as state affect, is used to define how an individual may feel for a few minutes or even an entire day. This can be compared to trait/dispositional affect, which is related to long-term personality characteristics that are evident regardless of context. When describing the results or details of other studies, the terms chosen by the authors of that particular study will be used.

While there are discrepancies in the emotion literature, in general, researchers appear to agree on the basic components of emotion. Feldman Barrett (1998) describes emotion as being made up of two components: valence and arousal. Valence refers to an individual's feeling of pleasantness or unpleasantness; in other words, PA or NA. Arousal refers to feeling activated or deactivated. Arousal involves the perceived activation, and sometimes physiological activation, associated with an individual's emotions. While emotions contain both valence and arousal components, researchers disagree on appropriate ways to measure and understand specific self-reported emotions.

One model of emotion that is consistent with the two component model discussed above places PA and NA on opposite poles of a single dimension. The circumplex model of affect describes emotions as being on an unpleasant-pleasant plane and a high-low activation plane. In this model, a variety of emotions are equally spaced around the two independent dimensions

(Lucas, Diener, & Larsen, 2003). Larsen and Diener (1992) have presented an example of a circumplex model of affect that was adapted from Russell (1980) and Watson and Tellegen (1985). While there is great disagreement regarding whether these dimensions are truly independent, this model is useful in that it allows us to map different feelings onto a precise coordinate of valence and activation. This may prove to be important in the context of health and pain, specifically. This generates the ability to compare different emotion types in a more precise manner. For example, while enthusiastic, cheerful and calm are all positive emotions, we can further divide them into activation levels (e.g., enthusiastic is high arousal, while calm would be unaroused). When affect is placed on a bipolar continuum (Russell, 1979) one cannot feel both PA and NA simultaneously. Further, this model suggests that PA and NA are not independent. Based on this assumption, any effect on one will have an influence on the other.

Instead of conceptualizing emotions as opposite poles of a single dimension, other researchers believe that pleasant and unpleasant affect are independent constructs. The Positive and Negative Affect Schedule, or PANAS, developed by Watson, Clark, and Tellegen (1988) measures positive and negative emotions via a multi-item adjective checklist. As in the previously mentioned model, the hierarchical structure of self-rated affect also has two independent dimensions. These dimensions consist of a high-low PA dimension and a high-low NA dimension.

The PANAS was created due to previous evidence highlighting the consistent emergence of PA and NA as independent in analyses of self-rated mood and “multidimensional scalings of facial expressions or mood terms” (Diener, Larsen, Levine, & Emmons, 1985; Russell, 1980, 1983; Stone, 1981; Watson, Clark, & Tellegen, 1984; Zevon & Tellegen, 1982, in Watson, Clark, & Tellegen, 1988, p. 1063). The model that emerged from Watson and colleagues’

research contains two higher order valence dimensions (PA and NA) that are composed of various lower level affective states reflecting the content of affect (Watson & Clark, 1994).

Watson and colleagues (1988) define PA within this model as the subjective feelings of enthusiasm, activity, and alertness. In the PA plane, high PA is defined as a state of high energy, whereas low PA is a state of sadness and lethargy (Watson, Clark, & Tellegen, 1988). The other dimension, NA, is defined by Watson and colleagues as subjective distress that includes in the high NA plane anger, disgust, and fear. Within both dimensions, activation is an implicit component at high and low levels of PA and NA (Feldman Barrett & Russell, 1999). However, there are many words not included in the PANAS that intuitively should be included, such as feeling calm or depressed. Without these words included in such a widely-used scale, researchers are left questioning what role these types of feelings have on various outcomes.

Research supporting the independence of PA and NA comes from low or non-significant correlations between PA and NA scales (Watson, Clark, & Tellegen, 1988). Watson & Clark (1994) have also found that PA could be increased through social interactions and exercise, while NA was not influenced by social interactions or exercise. Therefore, some researchers have concluded that PA and NA are independent components of affect and at the least, worth measuring separately and considering independently.

However, researchers promoting the bipolar pleasantness and activation model believe that their model is challenged due to faulty definitions and other methodological problems. For example, Feldman Barrett and Russell (1999) explain that NA and PA are occasionally found to have correlations of zero due to definitions that force them to remain uncorrelated. For example, in the development of the PANAS, words that would normally be highly inversely correlated in the NA and PA domains (e.g., happy versus unhappy) were left out intentionally to ensure

independence. We are then left with a scale which may show orthogonal constructs, but is absent in many key defining feelings of PA and NA (e.g., cheerful) and present in some less obvious emotions of PA and NA (e.g., attention). This scale can be compared to similar adjective checklists that include more obvious emotions of PA and NA such as the Profile of Mood States-Revised (POMS-R; McNair, Lorr, & Droppleman, 1992). The POMS uses terms from both valences and activation levels, such as sleepy, relaxed, on edge, and happy.

Instead of relying on a steadfast rule of bipolarity versus independence, it may be more important to consider the situations in which PA and NA are related. In one study by Diener and Emmons (1985), the researchers found that the relation between PA and NA depends on the time period being considered. PA and NA states are negatively related over short time spans because the two are unlikely to occur together at the same moment. As the time span increases, however, PA and NA became relatively independent. The researchers also found that there were strong negative correlations between PA and NA when an individual felt strongly emotional. These findings make intuitive sense given the multitude of positive and negative events, moods, and feelings that can occur over a year, therefore increasing independence, as opposed to how one feels at a given point in time. However, there are certain situations where in a given moment, certain positive and negative feelings can occur (e.g., Schadenfreude, crying at a happy occasion, etc.).

Other evidence supporting the idea that PA and NA are independent constructs comes from physiological research. PA may influence the opioid system through such means as exercise and laughter (e.g., Harte, Eifert, & Smith, 1995; Martin, 2002; Pedersen & Hoffman-Goetz, 2000; Wildmann, Kruger, Schmole, Niemann, & Matthaei, 1986) or through general emotional activation (Gerra et al., 1996, 1998), although research is still lacking at this point. PA

may also play a unique role in undoing the negative impact of NA, stress, and other negative experiences by reducing physiological activation levels to baseline (Fredrickson, Mancuso, Branigan, & Tugade, 2000). Although PA is a more likely pathway, stress has been shown to have some influence on endogenous opioids as well. For example, in healthy individuals, there is an increase in endorphin levels following a stressor (e.g., Beutler, Engle, Oro'Beutler, Daldrup, & Meredith, 1986; Colt, Wardlaw, & Frantz, 1981; Dubois et al., 1981; Naber et al., 1981), whereas in individuals with chronic stress, endorphin levels plateau at low levels in response to a stressor (Beutler, Engle, Oro'Beutler, Daldrup, & Meredith, 1986; Cohen, Cohen, Weingartner, Pickar, & Murphy, 1983).

Other negative emotions, on the other hand, seem to play a particularly important role in sympathetic activation and the hypothalamic-pituitary-adrenal (HPA) system (e.g., Ritchie & Nemeroff, 1991; Stokes, 1987; Villacres, Hollifield, Katon, & Wilkinson, 1987; Buchanan, al'Absi, & Lovallo, 1999; Glaser & Kiecolt-Glaser, 2005; Sirois & Burg, 2003). Finally, there is early evidence suggesting different patterns of brain activation for those with high trait PA versus NA (Davidson, 2004; LeDoux, 2000; Price, 2000; Vogt, 2005), not to mention the known brain areas associated with fear (i.e., amygdala) and other negative feelings that are not known to play a role in positive feelings.

Of the above physiological relations, the plausible connections of PA and endogenous opioids may be particularly important when considering pain outcomes. Endogenous opioids contribute in regulating the organism's response to physiological and environmental demands, including physical and psychological stress (Akil et al., 1984). These opioids have an analgesic function, which has been found to blunt the distressing affective component of pain without dulling the sensation itself (Drolet et al., 2001). Components of PA, particularly vigor and

excitement, may affect these opioid pathways. With this physiological evidence, the relationship between PA and pain provides a compelling area of research, especially because there are no known pathways between NA and opioid pathways.

NA, on the other hand, may influence health and pain in other ways. For example, there is evidence that people with state depression have suppressed immunity (Cohen & Rodriguez, 1995). In animal models, there is some evidence suggesting that this decreased immunity may lead to diseases that often have chronic pain as a core symptom (Amkraut, Solomon, & Kraemer, 1971; Beutler, Engle, Oro'Beutler, Daldrup, & Meredith, 1986; Rogers et al., 1980). Additionally, individuals high in NA report more symptoms when ill (Cohen et al., 1995; Cohen & Rodriguez, 1995) and report more pain. NA may exacerbate pre-existing pain, or it may cause people to attend to negative cues, leading to an increased awareness of pain.

Inflammation, mood, and pain. Another possible connection between PA, NA and pain is via inflammation of the immune system. Inflammation occurs when the body comes in contact with various stressors, which can be both of physical and psychological nature (Reiche, Nunes, & Morimoto, 2004). In particular, for tissue damage, the immune system initiates a host response to repair the damage. Various leukocytes, including neutrophils, monocytes, and eosinophils, attend to the damaged tissue and recruit other pro-inflammatory cells to the area to aide in the process (Coussens & Werb, 2002). Once at the site of tissue damage, the monocytes transform into macrophages, which produce growth factors and cytokines (Coussens & Werb, 2002). The cytokines are a particularly important component of the inflammation process because their main role is to regulate the function of immune cells. The pro-inflammatory cytokines IL-1, IL-6, and TNF essentially cause the inflammatory process. While this process is normal for a targeted tissue-damage response, chronic inflammation can occur and is suggested to be due to incessant

activation of the initiating factors or due to dysfunction in the cessation of the inflammation process (Coussens & Werb, 2002).

There appears to be a growing area of literature within the field of inflammation and mood, specifically regarding mood disorders such as depression. Research currently provides evidence that many people experiencing depression also have elevated levels of chronic inflammation (Raison, Capuron, & Miller, 2006). There is also some evidence that there is a positive association between depressive symptom severity and inflammatory markers (Alesci et al., 2005; Miller, Stetler, Carney, Freedland, & Banks, 2002; Raison et al., 2006; Thomas et al., 2005), and that individuals with minimal depression-related symptoms such as fatigue or insomnia also have heightened inflammatory responses (Irwin et al., 2004; Raison et al., 2006; Suarez, Lewis & Kuhn, 2002; Suarez, Lewis, Krishnan, & Young, 2004; van der Ven et al., 2003).

Additional evidence has found that the inflammatory process is also influenced through a PA pathway. One group of researchers exposed participants to rhinovirus or influenza and measured daily affect in addition to antibodies related to cytokines of interest (IL-6, IL-1 β , or TNF- α) over a period of six days (Janicki-Deverts, Cohen, Doyle, Turner, & Treanor, 2007). Participants who had been exposed to the virus produced greater levels of IL-6 when they were experiencing less PA. However, daily NA was not found to be related to daily cytokine levels. The researchers concluded that the hypothesis that inflammation results in greater NA was not supported by their data, indicating that there may be a specific connection between PA and health, independent of NA.

Similarly, the relationship between pain and inflammation is not yet understood. However, some animal research has been done investigating the maintenance of pain due to pro-

inflammatory cytokines. Specifically, Üçeyler and colleagues (2009) cite a number of studies indicating that pro-inflammatory cytokines exacerbate pain for three reasons: 1) peripheral neural tissue damage leads to persistent cytokine expression (Kleinschnitz, Brinkhoff, Zelenka, Sommer, & Stoll, 2004; Taskinen et al., 2000; Üçeyler, Tschärke, & Sommer, 2007), 2) exposing rats to pro-inflammatory cytokines invokes pain behavior (Junger & Sorkin, 2000; Schäfers, Sorkin, & Sommer, 2003; Zelenka, Schäfers, & Sommer, 2005), and 3) pain relief occurs after rats are given treatment containing anti-inflammatory cytokines or pro-inflammatory cytokine inhibitors (e.g. Vale et al., 2003; Wagner, Janjigian, & Myers, 1998).

Pain

Pain, especially persistent pain, is a complicated phenomenon that affects everyone at some point in their lifetime. While pain research has burgeoned recently, researchers began writing about pain over 60 years ago (Daniels, 1940). It has become such a popular topic due to pain's complexity; research focuses on the role emotional, cognitive, and social factors play in the development and maintenance of pain (Keefe et al., 2002). Additionally, because people vary in their responses to a painful condition (Keefe et al., 2002), it is imperative to understand why, how, and when people differ in order to prevent or diminish painful conditions. It can also not be ignored that pain is becoming a popular phenomenon to study due to increasing awareness of such pain conditions as fibromyalgia and rheumatoid arthritis, in addition to influence from the pain medication industry.

Gate-control model. Generally, pain is defined as an unpleasant, subjective “sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage” (Merskey, 1991 in Anand & Craig, 1996, p. 3). The physiological mechanisms

involved in the pain experience have been debated for over forty years, but Melzack and Wall's (1965) gate-control model appears to be the primary theory.

Pain receptors, or nociceptors, are connected to small nerve fibers that extend to the spinal cord. The spinal cord is also intersected by large nerve fibers that carry information unrelated to pain. As Melzack and Wall (1965) describe, the experience of pain depends on the ratio of stimulation being experienced by the small fibers and large fibers. If there is no stimulation, the brain is not receiving information related to pain, meaning that the gate is "closed." If the large fibers are being stimulated, the gate remains "closed" because those fibers excite the substantia gelatinosa, which inhibits the message being sent to the brain. However, if the small fibers receive more information, the first central transmission (T) cells are activated, meaning that the gate is "open" and pain is experienced.

Once the brain recognizes the pain stimulus, the individual's behavioral response to the painful stimulus can be observed. Melzack and Wall (1965) explain that once the brain recognizes the painful signal, the recognition is followed by "(i) a startle reflex, (ii) a flexion reflex, (iii) postural readjustment, (iv) vocalization, (v) orientation of the head and eyes to examine the damaged areas, etc." (p. 976).

In order to better understand pain and its relationship with emotion, researchers have used a number of methods. Most of these methods include pain induction in healthy samples of college undergraduates, or following pain reports of participants with chronic pain syndromes such as rheumatoid arthritis or fibromyalgia.

Pain induction studies. The first of these methods, pain induction, measures the participants' subjective pain reports after exposing them to a controlled pain stimulus such as cold temperature, electrical stimulation, or increased pressure (e.g., from a blood pressure cuff). In

order to evaluate whether mood influences pain reports, mood is often induced as well in a number of ways (guided imagery, film clips, etc.). In order to measure participants' subjective pain, researchers use terms such as pain tolerance time, pain intensity, and pain threshold. Pain tolerance time refers to the highest level of pain a participant can withstand, with longer times indicating higher pain tolerance. Pain intensity refers to the intensity of the painful stimulus that can be tolerated by the participant, with higher objective levels of intensity indicating higher pain tolerance. Lastly, pain threshold refers to when the participant first reports the stimulus as painful. By using these terms to measure pain tolerance, individual differences for a number of factors between participants can be compared.

When participants in pain induction studies are exposed to a variety of mood-inducing images, statements, or film clips, researchers have found that participants induced to a depressive or negative mood have decreased pain tolerance time whereas positive induction resulted in longer tolerance times (Alden, Dale, & DeGood, 2001; Zelman, Howland, Nichols, & Cleeland, 1991; Zillmann, De Wied, King-Jablonski, & Jenzowsky, 1996). Similarly, researchers using pain and mood induction methods have found that both attention and affect influence pain perception differently; affect appears to particularly influence pain unpleasantness while attention influences pain intensity (Kenntner-Mabiala, Andreastta, Wieser, Mühlberger, & Pauli, 2008). Specifically, the researchers found that exposure to negative pictures resulted in increased pain intensity and pain unpleasantness.

Another study measured whether certain cognitions influenced pain tolerance and intensity (Stevens, Pfost, & Heise, 1989). Participants were told to think about high pleasure (e.g., making love), low pleasure (e.g., cool breeze), high anger (e.g., familial conflict), or low anger (e.g., receiving a parking ticket) life experiences in order to evoke varied levels of intensity

in affect conditions. The participants were then exposed to a finger pressure stimulus. The researchers found that pleasant cognitions were related to higher tolerance for pain than the angry cognitions. Additionally, the intensity of the affect was not found to be a moderator between pain tolerance and pleasant cognitions.

Further research has been developed to measure whether positive emotions, particularly humor and relaxation, have an influence on pain tolerance and intensity. There has been evidence that being exposed to comedic films elevates the pain threshold (Zillmann, Rockwell, Schweitzer, & Sundar, 1993). To further evaluate whether this realm of PA actually influences pain reports, one group of researchers assigned participants to one of three humor groups (cheerfulness, exhilaration, and humor production) and collected baseline cheerfulness information to assign participants to groups of high or low trait-cheerfulness and high or low trait-seriousness (Zweyer, Velker, & Ruch, 2004). Participants then completed a cold pressor task to determine pain threshold, tolerance, and sensitivity. In order to measure genuine exhilaration, non-enjoyment, and laughter, participants were also evaluated based on their facial expressions using the Facial Action Coding System (FACS, Ekman & Friesen, 1978). The results provided evidence that pain thresholds were increased for all three humor groups and remained elevated for 20 minutes, but no differences were found between groups. An increase in the pain threshold was only found in participants who expressed overt facial enjoyment. Additionally, participants low in trait-seriousness had higher pain thresholds and pain tolerance than those participants high in trait-seriousness. This research indicates that disposition and naturally occurring emotion may be particularly important in understanding the influence of affect and pain.

Because of the positive results humor has shown in pain induction studies, and because of the logistical ease in implementing relaxation techniques with people experiencing pain, investigators have been interested in examining whether there is a difference between humor and relaxation in increasing pain tolerance in pain induction studies. One study found that discomfort thresholds were higher for participants in laughter and relaxation groups compared to participants in control and informative narrative groups (Cogan, Cogan, Waltz, & McCue, 1987). There was not a significant difference between the laughter and relaxation conditions, but the laughter condition appeared to have higher discomfort thresholds than the relaxation group. This would have been a useful analysis given that both interventions map onto different arousal levels. The question remains if high arousal PA or low arousal PA is more important for pain.

However, others have speculated that humor and relaxation are only effective due to the expectation that the methods should increase pain thresholds. In other words, the effects of humor and relaxation can be attributed to the placebo effect. To test this hypothesis, Mahony and colleagues (2001) assigned participants to either a humor or relaxation exposure condition where they were given one of two instructional sets meant to evoke the expectation that the condition was to increase or decrease discomfort thresholds for the pain induction (blood pressure cuff inflammation pressure). When the participants were told that the humor or relaxation would increase pain thresholds, the measured thresholds increased significantly. Similarly, when told that the humor or relaxation conditions would decrease pain thresholds, the measured thresholds decreased significantly. The investigators proposed that due to the results of this study, humor and laughter may only contribute to increased pain thresholds because of the expectation that the interventions would be successful. However, given that most studies make no specific suggestion that these interventions will alter their pain experience, and given the known logical

physiological pathways, while the placebo may play some role, it is unlikely to be entirely responsible.

While these cross-sectional experimental pain induction studies provide useful evidence in the relationship between temporary mood and pain reports, there are still gaps remaining in the literature. First, the method in general is a barrier to gain more information. With limited information from one time point, many assumptions must be made between the pain and mood induction. Additionally, the mood being induced in the laboratory is not naturally occurring, and it would be interesting to see how an individual would rate his or her pain ratings in an environment that allowed them to incorporate their natural mood state at that moment. We can look to research on participant samples with chronic pain conditions to evaluate natural mood at specific times during the day, and whether that mood is related to heightened or lowered pain discomfort due to pre-existing medical conditions.

Longitudinal studies. Weekly and daily diaries or interviews have been conducted with pain patients to gain a better understanding of the role that PA and NA play in their symptom report. Looking specifically at negative mood, some researchers have found that pain is triggered by negative mood in a sample of participants with Reflex Sympathetic Dystrophy Syndrome (Feldman, Schaffer-Neitz, & Downey, 1999). However, it is unclear which aspect of pain perception was affected by the participants' mood.

Another study assessed pain, stress, and mood in a daily diary with participants diagnosed with sickle-cell disease (Gil et al., 2004). The researchers found, using mood and stress as predictors of sickle-cell disease pain, and that higher levels of positive mood resulted in lower pain ratings on the same day and two days after the reported positive mood. Similarly, statistical analyses showed that higher levels of negative mood predicted increased pain ratings

the same day and two days later. Stress was also found to be associated with higher pain reports the same day and two days later. In this study, a number of control variables were used in order to prevent biased within-person predictors. Of these control variables, one of them included day-to-day levels of sickle-cell disease pain. These control variables were included based on evidence by Schwartz and Stone (1998) indicating that summing person means with the corresponding within-person diary predictors will lead to unbiased within-person predictor coefficients.

A similar study was conducted in rheumatoid arthritis patients, but the researchers looked at the intensities of the mood reported in addition to how PA and NA influenced pain reports by using the PANAS (Connelly et al., 2007). The investigators found relationships for both NA and PA: maintaining levels of NA from the individual's usual level and lowering NA from an elevated level the previous day predicted lower levels of reported pain. Similarly, maintaining PA and increasing it after a previous day's lowered levels predicted lower levels of reported pain.

It is clear at this point that there is remaining uncertainty regarding whether PA or NA has a more defined role in predicting pain (i.e., whether the effects are independent of one another and/or whether one is a stronger predictor of pain). However, more attention is now being focused on the potential of PA having a unique influence on predicting pain. First, it is important to decipher the relationship between PA and NA.

In one study conducted Potter, Zautra, and Reich (2000), weekly interviews were conducted to gain information related to affect (via the PANAS), stress (life events), and pain. The researchers found that there was a strong negative correlation between PA and NA during stressful weeks (i.e., weeks where multiple undesirable daily events occurred). Additionally, during stressful weeks, NA and pain were positively correlated. PA and pain were inversely

correlated during stressful weeks. However, during weeks without reported stress, while NA and pain were correlated, PA was uncorrelated with pain. The authors proposed the need to further investigate when and for whom PA and NA are related.

That very question was examined in the next study. Using participants with fibromyalgia or osteoarthritis, weekly interviews were conducted to measure weekly pain, affect, and interpersonal stress in addition to baseline measurements of personality traits and demographic data (Zautra, Johnson, & Davis, 2005). Specifically, information regarding PA and NA was collected on a weekly basis with the PANAS-X (Watson & Clark, 1999). Using multilevel modeling, it was found that on average, high NA was related to weeks with high pain, high interpersonal stress, and low PA. Also, overall increases in PA directly lowered NA and reduced the influence of high pain and high interpersonal stress on NA. In fact, the authors suggest that the data provide evidence that increases in PA lowers NA and blunts the impact of high pain and interpersonal stress on NA. An increase in NA during the previous week predicted greater pain during the current week, controlling for pain from the prior week. Similarly, participants with higher average levels of PA were less likely to show increases in pain throughout the weeks. Zautra and colleagues (2005) proposed that these results provide evidence that there is an indirect link between PA and pain disorders with NA acting as the mediator.

With more attention being concentrated on the influence of PA, Kratz, Davis, and Zautra (2007) used weekly interviews to measure pain acceptance (e.g., “It’s okay to experience pain”), pain severity, affect, and pain catastrophizing (e.g., how often the participant thinks regarding his/her pain that “It is terrible, and that it is never going to get any better”) in participants with osteoarthritis or fibromyalgia. Results indicated that pain acceptance was related to greater PA and unrelated to NA. It was also found that pain acceptance acted as a moderator in the

relationship between NA and pain, leading the researchers to propose that pain acceptance increases PA which thereby indirectly impacts NA. This study provided evidence that components of PA influence the experience of pain severity and loosen the connection between subjective pain reports and NA (Kratz, Davis, & Zautra, 2007). Supporting this idea, one study found that distress does not always accompany pain, and perhaps we should be looking in other directions (Hamilton, Zautra, & Reich, 2005).

Perhaps the strongest evidence for the influence PA has with pain is provided by a study by Strand et al. (2006). Using weekly phone contacts to interview participants with rheumatoid arthritis, information based on pain, affect, and interpersonal stress was collected. Results showed that NA correlated with weekly most intense pain and interpersonal stress, but NA did not correlate significantly with PA. Additionally, it was found that PA was an important buffer against NA when pain was reported to be most intense. The investigators concluded that PA may offer a resilience component within the pain and NA relationship due to an increase in subjective well-being, self-efficacy, and other coping mechanisms.

Ecological Momentary Assessment (EMA) studies. The previously discussed interview and diary methods were conducted once a day or on a weekly basis. This often leads to missing data and inaccurate retrospective accounts of the experienced pain and affect each day throughout the week. For example, extreme pain at the time of the report may influence the participant's ratings of the entire week's pain and affect. To avoid this problem, researchers in a number of areas are turning to Ecological Momentary Assessment (EMA), which involves using multiple assessments to capture real world experiences. One way researchers use EMA is through electronic data collection, which can send out reminders and collect the exact time each

participant enters his or her information on a handheld palm computer, such as a personal digital assistant (PDA).

Smyth and Stone (2003) provide three rationales for using EMA research. First, it prevents reporting bias. This is especially important for study protocols that require participants to recall details for a long duration of time. Events or mood states that occur at the moment of recall most readily come to mind and will bias the self-report (Smyth & Stone, 2003). Similarly, the use of EMA avoids “parking lot compliance,” which is the completion of diaries at the last minute while in the parking lot prior to an appointment. The software in PDAs allows researchers to track when the diary was completed.

EMA also increases the ecological validity of the findings because the participants are reporting information stemming from natural circumstances while in their natural environment. This is especially important for measuring constantly changing states such as mood or blood pressure (Smyth & Stone, 2003). Lastly, EMA provides researchers with temporal pattern of changes (Smyth & Stone, 2003). With this large amount of information, causal associations can be determined using statistical techniques such as multilevel modeling because the information is continuous rather than static.

Additionally, digital EMA increases compliance compared to typical diary studies. For example, Smyth and Stone (2003) found that in a study comparing paper diaries to PDAs, the paper diary group had a compliance of only 11-19%, whereas the compliance rates among the participants using PDAs were 94%. Not only does EMA increase the methodological robustness of a study, but it also provides the researcher with more data and better compliance rates among participants.

EMA was used by a group of researchers to evaluate the relationship between PA, NA, and pain in participants with fibromyalgia or arthritis (Zautra, Smith, Affleck, & Tennen, 2001). Information was collected at random as notified by the PDA three times a day. Like results have shown previously, the interaction between PA within-day and pain provided evidence that PA and NA had a stronger inverse relationship only when pain was high. While these findings replicate previous research, this study was informative because of the robust methodology allowing for accurate pain and affect accounts. One limitation of this study, however, is that there were no definitive answers regarding whether PA or NA predicted subjective pain reports.

Current Study

While all of the preceding studies provide valuable evidence to the relationship between affect and pain, there are remaining questions. First, the only longitudinal pain and affect data gathered have been in chronic pain samples. Little is known about the experience of general aches and pains in a healthy population and the role affect plays in the relationship. More information is also needed to evaluate if emotion only matters with chronic pain populations, or if the same theories can apply to healthy populations with more typical pains. Additionally, more information is needed using EMA to gather unbiased information multiple times each day. From this information, statistical analyses can be done to gain a better understanding of the relationship between PA and NA and subjective pain reports.

The current study seeks to improve the existing emotion literature by doing the following: 1) using digital EMA, giving more precise measurement, 2) using both PA and NA in order to understand their relative strength of association with pain, 3) examine this association over time in a longitudinal sample with two weeks of mood and pain data, and 4) investigate

naturally occurring pain and emotion in a healthy sample in an attempt to broaden the existing literature.

Moderators of the pain experience. A number of factors should be considered in analyzing the data from this study, particularly demographics, stress, depressive symptoms, quality of sleep, and trait affect. Stress influences pain as evidenced by research by Porter and colleagues (2000). The investigators found that in a sample of participants with sickle-cell disease, stress not only preceded days of high pain, but it was also a significant predictor of stress even after accounting for sickle-cell disease-related stressors (Porter et al., 2000). Other studies have also indicated the important role stress has in the relationship between mood and pain (Gil et al., 2004; Schwartz, Slater, & Birchler, 1994; Potter, Zautra, & Reich, 2000).

Depressive symptoms have also been found to be associated with pain reports. For example, in a review by Bair and colleagues (2003), the authors reported that those individuals with both depression and chronic pain report more intense and frequent pain, yet chronic pain populations also report experiencing more depressive symptoms. It could be that the two are interrelated, especially due to evidence of inflammation present in both depression and pain populations.

Pain reports are commonly associated with quality of sleep as well. One group of researchers found evidence that sleep quality provides a buffer against pain and PA and NA (Hamilton, Catley, & Karlson, 2007). Similarly, another group of researchers found that participants with fibromyalgia who were considered poorer sleepers via a sleep-quality interview also reported more pain (Affleck, Urrows, Tennen, Higgins, & Abeles, 1996). It appears that generally, sleep has an influence on affect, pain, or both (e.g., Graham & Streitel, 2010; O'Brien

et al., 2010), while the mechanisms are less clearly understood. Because of this, it is necessary to assess the influence of sleep on pain both across days and within day.

Finally, trait affect will also be analyzed as a moderator in this study due to the evidence that certain personality characteristics are associated with pain reports. Specifically, neuroticism has been found to be associated with increased pain reports. A study by Asghari and Nicholas (2006) found that in a sample of chronic pain patients, neuroticism was related to pain self-efficacy beliefs and pain control appraisals, indicating that individuals high in neuroticism have a difficult time adjusting to and dealing with pain.

It should be noted that these variables all have an affective component and influence pain. In other words, all of these variables are associated with mood variation and pain, whether it be chronic or laboratory induced. Including these variables, however, can provide a clearer picture of whether PA, PA's arousal levels, and NA can predict pain above what is already explained by these other factors.

Current hypotheses and analyses. Analyses will be completed to assess predictive power of PA and NA on within-day and next-day pain considering the influence of other critical variables.. It is important to examine the influence of affect both day-to-day and within day because these models allow for an evaluation of how affect over a longer period may differ from affect experienced at a particular moment.

Because of important covariates, the moderator of stress will be analyzed to determine if the interaction between stress and PA and stress and NA predict the experience of pain in this sample. An exploratory analysis will also be completed to determine which types of PA (high-arousal, or vigor, and low-arousal, or calm) are the strongest predictors of pain reports. Given the earlier discussion regarding valence differences within scales measuring emotion, investigating

the specific types of PA is essential in understanding what is most important in predicting pain levels. For example, interventions to help with pain (e.g., PA inducing) could be very different depending on the valence of the emotion (e.g., relaxation vs. humor). Studies have not looked at this, with the exception of the induction studies mentioned previously, so this study aims to uncover which types of arousal levels are connected to pain.

The hypotheses of this study include the following: 1) NA will be associated with higher within-day and next-day pain, even after accounting for other predictor variables, 2) PA will be associated with lower within-day and next-day pain, even after accounting for other predictor variables, 3) The PA/NA relationship with pain will be moderated by level of stress both within-day and across days, and 4) High-arousal PA and low-arousal PA will predict pain within-day and across days, even after accounting for other predictor variables.

Method

This investigation used an existing data set of 83 undergraduates at Carnegie Mellon University from a vaccination study. For detailed information about study protocol, please refer to the article by Pressman et al. (2005) and Miller et al. (2004).

Participants

Eighty-three college freshmen from Carnegie Mellon University participated in the study. All reported no chronic or acute illness, no regular medication regimen (with the exception of birth control), and good health upon entering the study. Participants were each paid \$120.

Design and Procedure

Baseline information regarding demographic, psychological, and general health practices was collected upon entering the study. The participants then used a PDA for 13 consecutive days (beginning two days after baseline assessment) in which they recorded their current emotions,

behaviors and symptoms four times a day at one, four, nine, and eleven hours after waking. Mood, stress, and pain were assessed at each assessment while health behaviors were assessed once a day. Participants were alerted by the PDAs when it was time to complete the questionnaires, and all information entered by the participants was time-stamped by the EMA software.

Materials

Depressive symptoms, affect, and psychological stress. The Center for Epidemiological Studies—Depression Scale (CESD-10; Andresen, Malmgren, Carter, & Patrick, 1994) was used to assess baseline symptoms of depression. The scale ranged from 0 to 3, with 0 indicating that the symptom occurred rarely or none of the time and 3 indicating that the symptom occurred most of the time. More symptoms of depression were indicated by higher scores ($\alpha = .79$).

PA and NA were measured at each alerted EMA entry by using a scale of 0 to 4 for each item, with 0 indicating that the item is not at all accurate to how the participant is feeling at that moment, and 4 indicating that the item is extremely accurate to how she/he is feeling. The adjective checklist used in this study was compiled by using emotion words derived from both the POMS and PANAS. Negative affect was measured based on two categories: anxiety (including jittery and nervous) and depression (including unhappy and sad). Positive affect was measured using three categories: vigor (including active, intense, lively, and enthusiastic), well-being (including happy and cheerful), and calm (including calm and relaxed). The categories for each affective domain were summed in order to create two separate dimensions for PA and NA. Over 13 days, the overall alpha for NA was .84-.91, while the overall alpha for PA was .86-.95. Average PA and NA scores were then generated for all 13 EMA days and yielded an NA alpha of .97 and a PA alpha of .94.

Psychological stress information was gathered by EMA at each time point with two items by asking the participants how overwhelmed and stressed they felt on a scale from 0 to 4, with 0 being not at all (stressed or overwhelmed) and 4 being extremely (stressed or overwhelmed). Average daily stress was then created by averaging the responses to both items each day.

Personality, self-esteem, and hostility scales. Neuroticism, extraversion, self-esteem, and hostility were measured at baseline for all participants. Neuroticism and extraversion were assessed using a modified version (see Feldman et al., 1999, for modifications) of Goldberg's Big Five Scale (10 items each; Goldberg, 1992). Participants responded to the items by thinking of how they generally feel on a scale from 0 to 4, 0 being not at all accurate and 4 being extremely accurate. Neuroticism and extraversion alphas were .84 and .92, respectively.

Self-esteem was assessed using the Rosenberg Self-Esteem Scale (Rosenberg, 1965). Participants responded to the items by judging the extent to which they agreed with each item on a scale of 1 to 4, with 1 being strong disagreement and 4 being strong agreement. The alpha for self-esteem was .91. Lastly, the Cook–Medley Hostility Scale (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989) was used to measure hostility. Participants answered 20 true/false questions, with the summation of the true responses equal to an overall hostility score. The hostility score alpha was .61.

Sleep. In order to gather information related to sleep, an abbreviated version of the Pittsburgh Sleep Quality Index (PSQI; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989) was used at baseline and one hour after waking during the 13-day data collection period. Sleep variables included sleep duration in hours, sleep loss in minutes, and sleep quality. From the eight questions in the measure, scores were rated on a scale from 1 to 4, with 1 indicating poor sleep and 4 indicating high quality sleep.

Pain. Pain was assessed at each time point across the 13 days using the Cohen-Hoberman Inventory of Physical Symptoms-Short Form (CHIPS-SF; Cohen, 1983). This scale assessed four general areas of pain, including 1) aches and pains, 2) muscle tension or soreness, 3) stomach upset or cramps, and 4) headache. Each item was rated on a 5-point scale, with 0 indicating that the participant was not bothered at all by a particular symptom, and 4 indicating that the participant was extremely bothered by the symptom. Average pain per time point and average daily pain ratings were created. All measures mentioned above are provided in the Appendix.

Statistical Procedure

The statistical analysis that was used in this study is multilevel modeling (MLM). To run the analyses, the PRELIS application from the LISREL software was used. This type of analysis is useful for data with complex patterns of variability, especially with nested sources of variability (Snijders & Bosker, 1999). Nested sources of variability can be best understood using an example referring to schools. For example, an example of nested, or clustered, data is one in which children are nested within schools, which are then nested within school districts. Because of the nature of the variables, a number of assumptions that can typically be held with ordinary data cannot be held when dealing with clustered data. For example, independence can no longer be assumed for each variable because cases within clusters are more similar than cases between clusters (Preacher, 2009). Because there is dependence among variables, this dependence can be evaluated (Snijders & Bosker, 1999).

Besides violating the independence assumption, using ordinary least squares (OLS) with clustered data would also violate assumptions of homoscedasticity and uncorrelated errors because sampling no longer adds unique information (Preacher, 2009). Ignoring these

dependency issues will yield misleading results, meaning that MLM is necessary to obtain unbiased results.

An additional advantage of MLM includes the ability to estimate the proportion of total variation in an outcome variable at a given level of the hierarchy (Hawkey, Preacher, & Cacioppo, 2007), which is why it is important to investigate the variables of interest across days and within each day, predicting pain at the next time point. Predictors are introduced into the model to explain variation at some of these levels. Similarly, given the longitudinal nature of the data, there is more latitude given to make causal statements in predicting the variable of interest, which is pain in this study.

Additionally, these analyses allow for the interpretation of both fixed and random effects. Interpreting fixed effects is fairly simple; a change in the predictor variable produces a change in the outcome variable, accounting for the other variables in the model. Random effects are a little more complicated; they address variability of an intercept, slope, and/or intercept-slope covariance. If an intercept variance is significant, it implies that people vary at the intercept; in other words, individuals differ at the value of pain where all other predictor variables equal zero. Random slopes indicate that the association between the predictor and the outcome variable differs across people. In other words, that the average effect of the predictor does not characterize all people. An example of this concept relevant to this study could be that the average effect of PA on pain does not fit in all cases. Finally, random intercept-slope covariances indicate whether there is an association between intercept location and steepness of the slope. For example, this value could suggest that people with higher levels at the intercept may have more gradual slopes, whereas those with lower levels at the intercept may have steeper slopes. A

hypothetical example relative to this study could be that those individuals with higher pain intercepts have a steeper slope given the NA predictor.

This study consists of two types of models: a two-level model and a three-level model. Predicting day-to-day pain is a two-level model, with the day (13) nested within participants (83). The information yielded from this model highlights how the influence of emotion on pain changes from day-to-day. Covariates can then be added in addition to the main predictor variables in order to examine whether the effect remains even when considering variables such as stress and sleep quality. By using MLM, the relationship between affect and pain will become clearer. Specifically, by using these sophisticated statistical procedures, a better understanding will be gained of whether PA or NA predicts pain over a period of days. In this particular model, day is considered a level-1 variable, and person is considered a level-2 variable.

Predicting pain within-day consists of a three-level model, with time points (4 times a day) nested within day (13 days) nested within participants (83). In this model, time point is the level-1 variable, day the level-2 variable, and person the level-3 variable. As mentioned above, predictor variables, such as PA and NA, can then be added to begin explaining variability in the random effects at lower levels (Hawkey, Preacher, & Cacioppo, 2007). Allowing for random effects, the associations between pain and NA, for example, may be stronger or weaker by day or person (at the second or third level). In particular, this model provides information regarding within-day variability in pain.

Results

Descriptive Statistics

Based on reports from the CHIPS-SF (Cohen, 1983), 95.1% of participants experienced pain at some point during the 13-day measurement period. Of the 83 participants who reported

pain, all scale possibilities were endorsed (0-4; $M = 1.189$, $SD = 1.177$). Participants (44.6% men) had a mean age of 18.29 years ($SD = .88$). A majority of the sample was Caucasian (63.3%) with the remaining participants (43.7%) endorsing other races.

The adjective checklist measuring affect was split into PA and NA, followed by a further division of PA in high-and low-activation items, specifically Vigor (high-arousal PA) and Calm (low-arousal PA). PA ($M = 1.650$, $SD = .841$), Vigor ($M = 1.322$, $SD = 1.011$), and Calm ($M = 1.863$, $SD = 1.048$) had a minimum of 0 and maximum of 4, while NA had less variability with a minimum of 0 and maximum of 3.5 ($M = .827$, $SD = .643$). PA, Vigor, and Calm all had normally distributed data at each time point and for each day. Only daily NA had slightly skewed data with a skewness statistic of 1.21 ($SE = .04$).

The two questions used at each time point to measure stress levels had a range from 0 to 4 endorsed ($M = 1.189$, $SD = 1.177$). Daily sleep quality was found to have a range of 1-4 ($M = 3.09$, $SD = .864$), with a majority of the participants endorsing high quality sleep (77.0% of responses were a 3 or 4). Finally, the baseline measure of neuroticism had a range of 0 to 25 ($M = 8.73$, $SD = 5.715$). Table 1 and Table 2 provide information regarding these descriptive statistics and sample characteristics.

Table 1
Sample Characteristics

Variable	<i>n</i>	%
Sex		
Male	37	44.6
Female	46	55.4
Race		
Caucasian	55	66.3
Non-Caucasian	28	43.7

Table 2
Descriptive Statistics

Measure	Minimum	Maximum	Mean	Standard deviation
Age	18	25	18.29	0.88
Pain				
CHIPS-SF	0	4	1.19	1.18
Affect				
PA	0	4	1.65	0.84
NA	0	3.5	0.83	0.64
Vigor	0	4	1.32	1.01
Calm	0	4	1.86	1.05
Stress	0	4	1.19	1.18
Sleep Quality	1	4	3.09	0.86
Neuroticism	0	25	8.73	5.72

Correlations were run between predictors used in the 2- and 3-level models, and all were found to be significant. However, this is not surprising because as mentioned earlier, the stress, sleep quality, and neuroticism variables all have affective components. The reason it is important to consider them all in the models is to understand how affect drives the model while accounting for these other important predictors, with an emphasis based on day-to-day and time-point variation. Predictor correlations for the 2- and 3-level models can be found in Table 3 and 4, respectively.

Table 3
2-Level Model Predictor Correlation Matrix

Variable	Sleep Quality	Neuroticism	Daily Stress	Daily PA	Daily NA	Daily Vigor	Daily Calm
Sleep Quality	---						
Neuroticism	-.21**	---					
Daily Stress	-.12**	.42**	---				
Daily PA	.31**	-.26**	-.24**	---			
Daily NA	-.17**	.52**	.68**	-.32**	---		
Daily Vigor	.25**	-.09**	-.07**	.83**	-.12**	---	
Daily Calm	.33**	-.37**	-.37**	.78**	-.48**	.39**	---

Note. **p < .01

Table 4
3-Level Model Predictor Correlation Matrix

Variable	Sleep Quality	Neuroticism	Time Stress	Time PA	Time NA	Time Vigor	Time Calm
Sleep Quality	---						
Neuroticism	-.21**	---					
Time Stress	-.10**	.37**	---				
Time PA	.25**	-.24**	-.30**	---			
Time NA	-.23**	.44**	.60**	-.47**	---		
Time Vigor	.19**	-.07**	-.10**	.82**	-.34**	---	
Time Calm	.24**	-.31**	-.37**	.71**	-.38**	.26**	---

Note. **p < .01

It should be noted that 29.1% of the daily sleep quality data were missing from the 83 participants in the study. Overall, 12.8% of the total data was missing from all variables included in the models. LISREL, the statistical software used in this study, handles missing data via full information maximum likelihood (FIML) estimation, which is the default for the program. It is important to note that listwise deletion was not utilized in the analyses. Additionally, an error structure was not defined during model development.

2- Level Model

Random effects models with covariates. An intraclass correlation (ICC) was conducted to determine if a multilevel modeling approach would be appropriate. Results from the ICC indicate that 60% of the observed variation in pain is due to differences among the participants. Due to the high percentage of variation in the level-2 variable, it is appropriate to use MLM to analyze the data.

To consider which predictor variables should be tested in various models, randomized effects models with covariates were done to determine which predictor variables were related to pain. These tests are conceptually similar to standard t-tests by examining mean differences, but t-tests differ due to the assumptions of independent observations and homoscedasticity, for example. Due to the large number of possible predictors, an alpha of .01 was used as a cut-off point to determine which variables would be kept to test in the models. The possible predictors included the following: age, gender, race, baseline sleep quality, baseline sleep efficiency, number of naps, baseline trait neuroticism, baseline trait extroversion, CESD score, time spent sleeping, time spent exercising, baseline trait hostility, baseline self-esteem, daily sleep quality, and daily stress. Of these variables, daily sleep quality, daily stress, baseline trait negative affect, cumulative CESD depression, and baseline trait neuroticism all had p-values equal to or less than .01 and were therefore tested in the models. As mentioned previously, it is important to note that while these predictors have affective components, they are being included in the model in order to provide evidence that specific affect (PA, NA, and the two activation components of PA) predicts pain day-to-day (for the 2-level model) and within day (for the 3-level model discussed later) on top of existing dispositional and behavioral differences.

Next, residual ICCs were conducted. The residual ICC estimates the proportion of the unexplained variance in pain due individual factors not included in the model. In other words, the residual ICC is an estimate of the remaining variability between people, controlling for covariates. The residual ICC values and values from random effects models can be found in Table 5. All values indicate that there is a large amount of variance accounted for by each predictor, so all were considered in the initial models.

Table 5
2-Level Model Predictor Testing

Predictor	Random Effects Models with Covariates			Residual ICC		
	<i>B</i>	Standard error	<i>p</i>	Level 2 <i>T</i>	Level 1 <i>T</i>	ICC
DSlpq	-0.044	0.018	0.016	0.103	0.043	0.706
negaff	0.013	0.003	0.000	0.080	0.066	0.550
CESD	0.017	0.006	0.008	0.090	0.066	0.578
neurot	0.024	0.006	0.000	0.080	0.066	0.548
bquality	-0.134	0.045	0.003	0.088	0.066	0.573
DPA	-0.121	0.021	0.000	0.093	0.064	0.592
DNA	0.073	0.015	0.000	0.091	0.065	0.583
DVigor	-0.098	0.015	0.000	0.096	0.063	0.604
DCalm	-0.047	0.017	0.005	0.093	0.066	0.585
Dstress	0.027	0.007	0.000	0.095	0.065	0.594

Model development. All final models are the result of using a tear-down model development approach. Using this approach, all possible predictors were included in the initial models and were later taken out of the model based on whether the fixed effects had a significant influence in the model. Therefore, not all significant predictors from the initial random effects models with covariates remained in the final models.

PA and pain. The final model includes the fixed effects daily positive affect (DPA), daily sleep quality (DSlpq), daily stress (DStress), and trait neuroticism (Neurot). CESD and trait negative affect were initially included in the model, but neither predictor predicted pain. The fixed effects for DPA ($B = -.072, p < .05$) and Neurot ($B = .023, p < .001$) were significant, meaning that both DPA and Neurot predicted next-day pain, independent of the other predictors in the model. Therefore, DPA predicted less next-day pain while neuroticism predicted more next-day pain.

DPA was included as a random effect. DSlpq was initially included as well, but it did not vary across people; therefore, it was not kept in the model. In other words, in the model of PA and pain, the variation of daily sleep quality in its association with pain across individuals does not vary and therefore should not be included in the model as a random effect. However, including DPA as a random effect yielded a significant effect at the intercept ($T = .226, p < .01$), indicating that individuals varied on how much they experienced PA each day. The model is provided in Table 6.

Table 6

2-Level Model: PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
DPA	-0.072	0.035	0.041
DSlpq	-0.034	0.018	0.062
DStress	-0.005	0.010	0.647
neurot	0.023	0.006	0.000

Slope	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.226	0.070	0.001
DPA/intercept	-0.058	0.030	0.052
DPA/DPA	0.014	0.013	0.279

NA and pain. The same predictors used in the model with PA and pain were found to be the best predictors for the NA and pain model as well, substituting daily negative affect (DNA) for DPA. The tear-down approach indicated that CESD and baseline negative affect should not be included in the model. The two significant fixed effects were DSlpq ($B = -.036, p < .05$) and Neurot ($B = .031, p < .001$), indicating that both variables were predictive of next-day pain, accounting for all other factors. High quality sleep predicted less next-day pain while high trait neuroticism predicted an increase in next-day pain.

DNA was included as a random effect, which yielded significant variability across the intercept ($T = .020, p < .05$) and slope steepness ($T = .021, p < .05$). This indicates that not only do individuals have differing levels of pain depending on their level of NA, but these differing

initial levels of DNA at the intercept influence the steepness of the slope in relation to next-day pain. However, because there was not a significant fixed effect with DNA ($B = .044, p = .183$) this model indicates that the best predictor of pain is neuroticism. The model is provided in Table 7.

Table 7

2-Level Model: NA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
DNA	0.044	0.033	0.183
DSlpq	-0.036	0.018	0.044
DStress	-0.013	0.012	0.280
neurot	0.030	0.006	0.000

Slope	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.020	0.010	0.038
DNA/intercept	0.021	0.008	0.012
DNA/DNA	0.009	0.009	0.287

High-arousal PA and pain. The same three predictors were used in this model as in the PA and NA models, in addition to daily vigor (DVigor) rather than DPA and DNA. Once again, CESD and baseline trait negative affect were not included in the model due to their lack of influence in the model. The only significant fixed effect was Neurot ($B = .024, p < .001$), indicating that the best predictor of next-day pain within this model is neuroticism; high trait neuroticism is positively associated with higher next-day pain.

DVigor and DSlpq were both included in the model as random effects. It is important to note that keeping both DVigor and DSlpq in the model as random effects resulted in nonsignificance in the fixed effects for the predictor DVigor ($B = -.054, p = .066$). However, it was judged to keep both DSlpq and DVigor as random effects because of the intriguing results of significant variability across people, indicating that DSlpq and DVigor seem to have more variability within-person than between-people ($T = .010, p < .05$; $T = .027, p < .01$, respectively). Table 8 contains the high-arousal PA model.

Table 8

2-Level Model: High-Arousal PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
DVigor	-0.054	0.029	0.066
DSlpq	-0.034	0.021	0.111
DStress	-0.002	0.010	0.874
neurot	0.024	0.006	0.000

Slope	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.213	0.071	0.003
DVigor/intercept	-0.030	0.021	0.146
DVigor/DVigor	0.027	0.010	0.007
DSlpq/intercept	-0.019	0.016	0.233
DSlpq/DVigor	-0.011	0.006	0.056
DSlpq/DSlpq	0.010	0.005	0.041

Because of the strong person-level effects of the DSlpq predictor, DSlpq was further separated into a level-1 and level-2 variable to determine how much variance in pain is due to within-person variation (via level-1 fixed and random effects) and between-person variation

(level- 2 fixed effect). By incorporating the same predictors with the exception of splitting the DS1pq predictor into two variables (level-1 and level-2), and by keeping DVigor and the level-1 sleep quality variable as random effects, it is shown by the model that DVigor ($B = -.027, p < .01$), Neurot ($B = .027, p < .01$) and the level-2 sleep quality variable ($B = -.300, p < .001$) were significant fixed effects, with daily vigor negatively associated with next day pain, high trait neuroticism predictive of higher next-day pain and between-person differences in quality sleep predictive of lower next-day pain.

The random effects in the model were significant at the person-level for both DVigor ($T = .476, p < .001$) and the level-1 sleep quality variable ($T = .061, p < .001$). Taken together with the fixed effects, these results provide evidence that daily vigor and the level-1 sleep quality predictor vary at the person level, but this is less interesting because the average effect is not significant. Daily vigor, neuroticism and the level-2 sleep quality variables are most predictive of pain in this model, which is provided in Table 9.

Table 9

2-Level Model: High-Arousal PA and Pain with Separated Sleep Quality

Fixed Effects			
Predictor	<i>B</i>	Standard error	<i>p</i>
DVigor	-0.027	0.316	0.001
DSlpq_L1	-0.010	0.032	0.761
DSlpq_L2	-0.300	0.086	0.000
DStress	0.006	0.004	0.138
neurot	0.027	0.008	0.001
Random Effects			
Slope	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.891	0.147	0.000
DVigor/intercept	-0.588	0.103	0.000
DVigor/DVigor	0.476	0.079	0.000
DSlpq_L1/intercept	-0.005	0.031	0.873
DSlpq_L1/DVigor	0.009	0.023	0.699
DSlpq_L1/DSlpq_L1	0.061	0.012	0.000

Low-arousal PA and pain. The same three predictors were used in this model as the above vigor model, substituting daily calm (DCalm) for DVigor. CESD and baseline trait negative affect were not included in the model due to lack of effects. No fixed effect was found for DCalm; the only significant fixed effect was a positive association between Neurot and pain ($B = .026, p < .001$), indicating that neuroticism was the best predictor of next-day pain within the low-arousal PA model.

DCalm was included as a random effect, with the only significant variability occurring at the intercept ($T = .134, p < .01$). In this model, daily levels of feeling calm are not associated with next-day pain; instead, the model is being driven by neuroticism. The model for low-arousal PA and pain are provided in Table 10.

Table 10

2-Level Model: Low-Arousal PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
DCalm	-0.034	0.030	0.257
DSlpq	-0.035	0.019	0.061
DStress	-0.006	0.012	0.630
neurot	0.026	0.007	0.000

Slope	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.134	0.048	0.005
DCalm/intercept	-0.008	0.018	0.657
DCalm/DCalm	-0.007	0.007	0.368

Stress moderator for PA and pain. The interaction term DPA*DStress was included in the model for PA that also included the fixed effects DPA, DSlpq, DStress, and Neurot. CESD and baseline trait negative affect were tested, but not ultimately included in the model due to a lack of influence in the model. The interaction was found to be nonsignificant ($B = -.001$, $p = .953$). Table 11 provides the model for testing stress as a moderator to PA and pain.

Table 11

2-Level Model: Stress as a Moderator of PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
DPA	-0.068	0.049	0.165
DSlpq	-0.034	0.018	0.067
DStress	-0.006	0.023	0.802
neurot	0.025	0.006	0.000
DPA*DStress	-0.001	0.013	0.953

Stress moderator for NA and pain. The interaction term DNA*DStress was included in the model for NA that included the fixed effects DNA, DSlpq, DStress, and Neurot. Again, due to lack of significance, CESD and baseline trait negative affect were not included. The interaction was found to be nonsignificant ($B = -.018, p = .054$). This model is provided in Table 12.

Table 12

2-Level Model: Stress as a Moderator of NA and Pain

Predictor	<i>B</i>	Fixed Effects	
		Standard error	<i>p</i>
DNA	0.113	0.046	0.014
DSlpq	-0.040	0.018	0.028
DStress	0.005	0.017	0.779
neurot	0.024	0.006	0.000
DNA*DStress	-0.018	0.009	0.054

3-Level Model

Random effects models with covariates. Similar to the 2-level models, ICCs were conducted to determine if a multilevel modeling approach would be appropriate. Results from the ICC indicate that 36.6%, 19.4%, and 43.0% of the variation in pain is due to differences among the time points (level-1), day (level-2), and individual (level-3), respectively. Due to the variation among the three levels, it is appropriate to use MLM to analyze the data.

Again, in order to consider which predictor variables should be tested in various models, random effects models with covariates were completed to determine which predictor variables were related to pain. Similar to the 2-level model, an alpha of .01 was used as a cut-off point to determine which variables would be kept to test in the model. The list of variables used in the 2-level model was also tested in the 3-level model, in addition to stress at each time point. Of these variables, the same predictors found to be significant in the 2-level model were also significant in the 3-level model. These predictors include daily sleep quality, baseline trait negative affect,

cumulative CESD depression, and baseline trait neuroticism. Stress at each time point was significant as well.

Next, residual ICCs were conducted. The residual ICC values and random effects models values can be found in Table 13. All values indicate that there is a large amount of variance

Table 13
3-Level Model Predictor
Testing

Predictor	Random Effects Models with Covariates			Residual ICC					
	<i>B</i>	Standard error	<i>p</i>	Level 3 <i>T</i>	Level 2 <i>T</i>	Level 1 <i>T</i>	ICC (1)	ICC (2)	ICC (3)
DSlpg	-0.047	0.018	0.010	0.102	0.025	0.068	0.346	0.130	0.524
negaff	0.013	0.003	0.000	0.080	0.043	0.081	0.398	0.212	0.390
CESD	0.018	0.006	0.007	0.089	0.043	0.081	0.380	0.202	0.417
neurot	0.024	0.006	0.000	0.079	0.043	0.081	0.399	0.213	0.388
bquality	-0.132	0.044	0.003	0.088	0.043	0.081	0.383	0.204	0.413
TPA	-0.088	0.009	0.000	0.092	0.042	0.080	0.374	0.196	0.430
TNA	0.137	0.011	0.000	0.087	0.040	0.079	0.383	0.194	0.422
TVigor	-0.046	0.007	0.000	0.096	0.042	0.081	0.370	0.192	0.438
TCalm	-0.044	0.007	0.000	0.092	0.044	0.080	0.370	0.204	0.426
TStress	0.040	0.007	0.000	0.094	0.042	0.081	0.371	0.195	0.434

accounted for by each predictor at each level, so all were considered in the initial models.

Model development. As in the 2-level model development process, all final models are the result of using a tear-down model development approach. Therefore, not all significant predictors from the random effects models with covariates remained in the final models. Specifically, CESD and baseline trait negative affect were tested in each model, but were not included in the final models due to the lack of significant fixed effects within each model.

PA and pain. The final model includes the fixed effects of positive affect at each time point (TPA), daily sleep quality (DSlpq), stress at each time point (TStress), and trait neuroticism (Neurot). The fixed effects for TPA ($B = -.049, p < .01$), DSlpq ($B = -.038, p < .05$) and Neurot ($B = .025, p < .001$) were significant, meaning that positive affect at each time point, daily sleep quality, and trait neuroticism predicted pain within-day, independent of the other predictors in the model. Specifically, higher PA at a certain time point was negatively related to lower pain later in the day, high quality sleep was negatively related to less pain later in the day, and trait neuroticism positively predicted high pain levels later in the day.

TPA was included as a random effect at the level-1 slope due to its measurement at each time point. DSlpq was initially included as well as a level-2 random effect, but it did not vary across people. Including TPA as a random predictor yielded significant effects at the intercept ($T = .093, p < .001$), indicating that individuals differ in the level of pain when all predictors equal zero. This model is provided in Table 14.

Table 14

3-Level Model: PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
TPA	-0.049	0.017	0.004
neurot	0.025	0.006	0.000
DSlpq	-0.038	0.019	0.043
TStress	-0.011	0.014	0.441
Level 1 Slopes	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.093	0.014	0.000
TPA/intercept	-0.004	0.008	0.599

TPA/TPA	-0.003	0.004	0.384
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NA and pain. The same predictors used in the model with PA and pain were found to be the best predictors for the NA and pain model as well, substituting NA at each time point (TNA) for TPA. TNA ($B = .092, p < .001$) and Neurot ($B = .023, p < .001$) were significant fixed effects in the model, meaning that both NA and neuroticism were positively associated with pain, and that they are the best predictors for within-day pain, accounting for all other predictors in the model.

TNA was included as a random effect as well, which yielded a significant effect at the intercept ($T = .007, p < .05$) and in the steepness of the slope ($T = .031, p < .001$). This indicates that different initial levels of TNA (at the intercept) influence the steepness of the slope in relation to pain at each time point. Table 15 provides the 3-level model for NA and pain.

Table 15

3-Level Model: NA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
TNA	0.092	0.022	0.000
neurot	0.025	0.006	0.000
DSlpq	-0.029	0.017	0.094
TStress	-0.017	0.013	0.185
Level 1 Slopes	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.007	0.003	0.013
TNA/intercept	0.031	0.006	0.000
TNA/TNA	0.009	0.009	0.331

High-arousal PA and pain. Once again, DSlpq, Neurot, and TStress, in addition to vigor at each time point (TVigor), were the predictors in this model. Significant fixed effects included TVigor ($B = -.025, p < .05$), DSlpq ($B = -.036, p < .05$), and Neurot ($\beta = .026, p < .001$). High-arousal PA at a given time point, daily sleep quality, and trait neuroticism were the best predictors of within-day pain, accounting for all other predictors. Both TVigor and DSlpq were negatively associated with pain, indicating that higher levels of these variables were associated with lower levels of pain later in the day. On the other hand, higher Neurot was positively related to pain, indicating that higher levels of trait neuroticism were associated with higher levels of within-day pain.

TVigor was included in the model as a random effect, and a significant effect was found across all slopes, including at the intercept ($T = .112, p < .001$), the slope steepness ($T = -.031, p < .001$), and the variance across individuals ($T = .014, p < .001$). These data indicate that the difference in initial levels of pain based on level of vigor at each time point influence the steepness of the slope between vigor and pain. Also, the average association between vigor and pain does not fit across individuals in this model, as supported by the significance in the slope random effect. This model is provided in Table 16.

Table 16

3-Level Model: High-Arousal PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
TVigor	-0.025	0.012	0.032
neurot	0.026	0.006	0.000
DSlpq	-0.036	0.018	0.044
TStress	-0.008	0.012	0.504

Level 1 Slopes	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.112	0.010	0.000
TVigor/intercept	-0.031	0.006	0.000
TVigor/TVigor	0.014	0.003	0.000

Low-arousal PA and pain. The same three predictors were used in this model as in the high-arousal model, substituting calm at each time point (DCalm) for TVigor. Significant fixed effects were once again TCalm ($B = -.032, p < .01$), DSlpq ($B = -.038, p < .05$), and Neurot ($B = -.025, p < .001$). Similar to the high-arousal PA model, this model indicates that low-arousal PA at a given time point, daily sleep quality, and trait neuroticism were the best predictors of within-day pain, accounting for all other predictors. Once again, TCalm and DSlpq were negatively associated with within-day pain, while Neurot was positively associated with within day pain.

TCalm was included as a random effect, and the variability at the intercept and steepness of the slope was significant ($T = .110, p < .001$; $T = -.016, p < .01$, respectively), indicating that

the slope steepness in the association between calm and pain varies depending on the level of pain at the intercept. This model is shown in Table 17.

Table 17

3-Level Model: Low-Arousal PA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
TCalm	-0.032	0.011	0.003
neurot	0.025	0.006	0.000
DSlpq	-0.038	0.018	0.034
TStress	-0.007	0.014	0.606
Level 1 Slopes	Random Effects		
	<i>T</i>	Standard error	<i>p</i>
intercept/intercept	0.110	0.012	0.000
TCalm/intercept	-0.016	0.005	0.002
TCalm/TCalm	0.003	0.002	0.115

Stress moderator for PA and pain. The interaction term TPA*TStress was included in the model for PA that included the fixed effects TPA, DSlpq, TStress, and Neurot. The interaction was found to be nonsignificant ($B = -.015, p = .138$). This model is provided in Table 18.

Table 18

3-Level Model: Stress as a Moderator of PA and Pain

Predictor	<i>B</i>	Fixed Effects	
		Standard	
		error	<i>p</i>
TPA	-0.032	0.020	0.100
neurot	0.025	0.006	0.000
DSlpq	-0.037	0.018	0.038
TStress	0.011	0.019	0.575
TPA*TStress	-0.015	0.010	0.138

Stress moderator for NA and pain. The interaction term TNA*TStress was included in the model for NA that included the fixed effects TNA, DSlpq, TStress, and Neurot. The interaction was found to be nonsignificant ($B = -.007$, $p = .593$). Table 19 provides the model for testing the interaction between stress, NA, and pain.

Table 19

3-Level Model: Stress as a Moderator of NA and Pain

Predictor	Fixed Effects		
	<i>B</i>	Standard error	<i>p</i>
TNA	0.108	0.031	0.000
neurot	0.023	0.006	0.000
DSlpq	-0.038	0.018	0.035
TStress	-0.012	0.019	0.518
TNA*TStress	-0.007	0.013	0.593

Discussion

Depending on the model, the time point (3-level) and day-to-day (2-level) models have somewhat different findings, indicating meaningful differences between pain and mood depending on when each is being assessed. The day-to-day model indicated that PA (but not NA) can predict next-day pain. This lack of association may be attributable to the consistent effects of trait neuroticism on next-day pain, which is consistent with past research showing that this is an important variable in reports of pain and other symptoms (Affleck, Urrows, Tennen, & Higgins, 1992; Asghari & Nicholas, 2006; DeGood, 2000; Katon, Unutzer, & Russo, 2010; Roelofs, Huibers, Peeters, & Arntz, 2008).

In the within-day time model, however, PA, NA, high-arousal PA, and low-arousal PA were all found to significantly predict within-day pain. Additionally, depending on the model,

sleep quality, and neuroticism were also effective in predicting within-day pain, independent of other predictors.

Finally, the hypothesis that stress would moderate the relationship between PA and pain and NA and pain was not supported in either the 3-level and 2-level model.

PA v. NA

Between-day. In considering the findings from both the 2-level and 3-level models, it can be concluded that PA is more predictive of pain than NA when looking across days and controlling for the relevant pain-associated trait and behavioral measures. This may be due to the strong effects of baseline dispositional NA, neuroticism, etc. on pain and symptom report obscuring the variable NA measures, or, alternatively, these dispositional NA characteristics may simply be more important for pain than transient experiences of negative mood. Dispositional PA, on the other hand, was not correlated with pain reports, but was on a day-to-day- level. The finding that dispositional PA was not associated with pain is counter to what other investigators have found in previous research (e.g., Guadagnoli & Mor, 1989; Potter, Zautra, & Reich, 2000). However, a possible explanation for this is that pain at a particular moment changes one's perceptions of PA (Pressman & Cohen, 2005). This particular momentary experience of pain may be more applicable to these general aches and pains in a healthy sample rather than the sometimes intense pain experienced by chronic pain samples.

There are a few other possibilities to describe this discrepancy as well. One explanation could be that state/transient NA is less predictive than trait NA. In other words, state NA may not have had a significant effect day-to-day because being more negative in general (trait NA) is more predictive compared to the variation in NA day-to-day. The effect of neuroticism has been supported by previous research demonstrating that individuals higher in trait neuroticism also

report more pain (e.g. Affleck, Urrows, Tennen, & Higgins, 1992; Asghari & Nicholas, 2006; DeGood, 2000; Katon, Unutzer, & Russo, 2010).

Another reason PA and pain may be specifically linked is the physiological pathways between positive mood and health outcomes. PA has a suggested influence on the opioid system (e.g., Gerra et al., 1996, 1998; Harte, Eifert, & Smith, 1995; Martin, 2002; Pedersen & Hoffman-Goetz, 2000; Pressman & Cohen, 2005; Wildmann, Kruger, Schmole, Niemann, & Matthaei, 1986). The influence of PA on the opioid system may be especially important in the relationship between PA and pain, because the endogenous opioids contribute to the analgesic function associated with blunting the affective experience of pain (Drolet et al., 2001). Finally, PA may have an important role in influencing the immune system (Janicki-Deverts, Cohen, Doyle, Turner, & Treanor, 2007), therefore having an influence on the organic experience of pain. In one study, PA was tied to the more accurate perception of an objective cold, whereas NA was correlated only with believing that one had a cold (Cohen, Alper, Doyle, Treanor, & Turner, 2006). It may be that in healthy individuals, this stronger correlation pervades where PA is objectively predicting lower pain in the future whereas NA is simply predicting feeling poorly at that moment.

Within-day. Predicting pain within-day yielded different results. The simultaneous effects of PA and NA at a single time point were predictive of pain with PA associated with less pain and NA associated with increased pain. This difference may be due to the differential times on the measures. At a given time point, it may be that the affective state has a critical impact on how one feels, and therefore reflects on the somatic experiences. Therefore, pain may be more related to how an individual “feels” than strict PA or NA. In other words, at a given moment, if an

individual is experiencing pain, he or she will endorse NA, whereas if he or she is not experiencing pain, PA will be endorsed.

The data from this study seem to indicate that the effects of PA and NA are contingent on the timing of measurement. This supports findings by Diener and Emmons (1985) that PA and NA will be related depending on the time period mood is measured in addition to the strength of a given emotion. Over short time spans (for example, within-day measurement of mood), PA and NA have a negative association. However, over a longer time span (for example, over a number of days), the negative association between PA and NA weakens and the relationship becomes more independent. There is no surprise, then, that both PA and NA predict pain within-day, because at any given moment in time, these two variables are highly correlated. When we move to a broader period, these variables are less strongly tied, and only PA predicts pain day-to-day.

Stress and Sleep

Stress was not a significant predictor of pain when included in these models due to the influence of affect on predicting next-day and within-day pain. Even so, stress was consistently included in the models due to previous research findings suggesting that stress often moderates the pathway between mood and pain. Specifically, investigators have shown that PA is predictive of pain in chronic pain patients when stress is accounted for (Potter, Zautra, and Reich, 2000; Zautra, Johnson, & Davis, 2005). Additionally, it is known that stress can have a significant effect on health by impacting the immune system (Taylor, 2006). Based on the findings from this study and accounting for information from previous research, it seems that the role of stress in the relationship between mood and pain is less important than the impact of mood directly on pain from day-to-day or within-day measures of pain.

Similarly, sleep quality was included in all of the models based on previous research that sleep has an impact on mood, pain, and general health (e.g. Affleck, Urrows, Tennen, Higgins, & Abeles, 1996; Graham, J. E., & Streitel, K. L., 2010; Hamilton, Catley, & Karlson, 2007; O'Brien et al., 2010). The data indicate that sleep quality was predictive of pain within the daily NA model, within-day PA model, within-day high-arousal PA model, and within-day low-arousal PA model. Therefore, the results from this study support previous findings that sleep quality predicts next-day and within-day pain in certain models. Sleep does in fact have an association with pain, but this association is not entirely due to the effect that sleep quality has on mood. In fact, the effect of sleep on pain was independent of mood.

Components of PA

The two components of PA tested in this study were high-arousal PA (Vigor) and low-arousal PA (Calm). For the 2-level day-to-day model, both high-arousal and low-arousal PA did not predict next-day pain. However, both high-arousal and low-arousal PA did predict within-day pain in the 3-level model. Therefore, the effects of high- and low-arousal PA are important at a given moment rather than over a period of days. This could be due to the physiological pathways associated with high- and low-arousal PA; the effect only persists at the moments those pathways are being influenced.

The finding of high-arousal PA predicting pain within-day lends support to its influence on health outcomes, specifically pain. It has been suggested that the high-arousal component of PA specifically can influence the endogenous opioid pathways, thereby diminishing the pain experience (Drolet et al., 2001). It appears that the role of high-arousal PA is especially important in reducing pain at a particular moment rather than over time. This may be due to

physiological mechanisms, but may also be due to simple attention differences and how high energy may divert attention away from symptoms (Jensen & Karoly, 1991).

The finding of low-arousal PA predicting pain within-day is consistent with interventions using relaxation techniques in chronic pain patients, especially within the domain of mindfulness-based interventions. The basic principles of mindfulness have evolved from Eastern meditation practices, and focus on bringing an individual's attention into the present moment (Carlson & Speca, 2007). Mindfulness interventions have been found to be successful at improving pain, sleep, and fatigue in a number of chronically ill populations (e.g. Carlson & Garland, 2005; Kabat-Zinn, 1982; Reibel, Greenson, Brainard, & Rosenzweig, 2001). Therefore, it is no surprise that low-arousal PA predicted within-day pain in this study. As shown in these findings, it is supported that low-arousal PA has an effect on pain in the moment, but it does not necessarily last over time (day-to-day).

Limitations

Because this study was based on a study that had already been conducted, there are a number of limitations. First, because the previous study had different primary hypotheses, and because the pain questionnaire was not the primary outcome variable of interest in the original study, it could not be modified for this study. It may have been useful to analyze the specific kinds of pain the participants were experiencing at each time point. It would have been equally beneficial to gather qualitative data on the participants' beliefs about the impetus of the pain.

Another limitation with this study is that a large percentage of the daily sleep quality data was missing. As a result, it is possible that the lack of data is influencing the small effects found within the models for the sleep quality predictor. Therefore, some of the results based on sleep quality should be interpreted with caution. However, the results found were in a direction that

was consistent with previous research. Based on this, it appears that the daily sleep quality predictor used in the models was able to accurately predict pain.

Future Directions

This study should be replicated with specific aims that focus on the questions posed in this manuscript. By doing this, mechanisms within PA and NA can be further investigated. For example, high-arousal PA specifically should be investigated to better understand the underlying mechanisms between vigor and health outcomes. This can be done in both a healthy and chronically ill population. Also, an interesting direction this research could take would be collecting markers of physiological functioning such as immune markers or salivary cortisol. By collecting this data, it can be better understood how PA, and components of PA, predict pain and body functioning in general.

Finally, these results support the use of positive psychology interventions to cope with particular types of pain, and can be applied to populations with chronic pain conditions. Some positive psychology interventions have already shown success in chronic pain patients. For example, positive psychology-based mindfulness has been used with cancer patients. In these studies, the cancer patients not only reported improved pain, but also less fatigue and improved sleep and general quality of life (Carlson and Garland, 2005; Lengacher et al., 2009; Reibel, Greenon, Brainard, & Rosenzweig, 2001).

Expressive writing interventions, specifically, benefit-finding interventions, have also shown improved physical functioning as evidenced by less physical symptom ratings in cancer patients participating in such interventions (Burton & King, 2004; Burton & King, 2008; Stanton et al., 2002).

Based on the results of this study, PA is a better predictor of next-day pain than NA. When assessing pain within-day, PA, NA, high-arousal PA, and low-arousal PA are all able to accurately predict pain. However, neuroticism and sleep quality have important implications in predicting pain as well, and the role of stress in predicting pain still needs to be better understood. Focusing on the positive instead of the negative allows advancement in the knowledge of not only what affects pain, but how to deal with it as well.

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Appendix A

Background Questions

1. How old are you? _____ years
2. Are you male or female? _____ male _____ female
3. How would you describe your primary racial or ethnic group?
 - _____ (1) White, Caucasian
 - _____ (2) Black, African-American
 - _____ (3) Native American, Eskimo, Aleut
 - _____ (4) Asian or Pacific Islander
 - _____ (5) Hispanic, Latino
 - _____ (6) Other specify _____

Appendix B

CESD-10

Instructions: Please read a list of the ways you may have felt or behaved recently. For each statement, please indicate how often you have felt this way during the past week.

1. I was bothered by things that don't usually bother me.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

2. I had trouble keeping my mind on what I was doing.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

3. I felt depressed.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

4. I felt that everything I did was an effort.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

5. I felt hopeful about the future.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

6. I felt fearful.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

7. My sleep was restless.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

8. I was happy.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

9. I felt lonely.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

10. I could not get “going”.

___0=rarely or none of the time ___1=some of the time ___2=occasionally ___3=most of the time
(less than 1 day) (1-2 days) (3-4 days) (5-7 days)

Appendix C

Emotion and Stress Adjective Checklist

Affect Adjectives	Diener & Larson Circumplex	Ursula & Hertzog POMS	PANAS
Quiet	Low activation		
Passive	Low activation		
Jittery	Activated Unpleasant	Anxiety	
Nervous	Activated Unpleasant	Anxiety	Negative
Unhappy	Unpleasant	Depression	
Sad	Unpleasant	Depression	
Happy	Pleasant	Well-being	
Cheerful	Pleasant	Well-being	
Drowsy	Unactivated Unpleasant	Fatigue	
Tired	Unactivated Unpleasant	Fatigue	
Relaxed	Unactivated Pleasant	Calm	
Calm	Unactivated Pleasant	Calm	
Active	High Activation	Vigor	Positive
Intense	High Activation	Vigor	
Lively	Activated Pleasant	Vigor	
Enthusiastic	Activated Pleasant	Vigor	Positive

<i>OTHER ADJECTIVES</i>	
Loneliness	Stress
Isolated	Overwhelmed
Lonely	Stressed

Appendix D

G-NE

Instructions: Below is a list of common human traits. For each trait, circle the response that best indicates how accurately that trait describes you. Describe yourself as you see yourself at the present time, not as you wish to be in the future. Describe yourself as you are generally or typically, as compared with other persons you know of the same sex and roughly the same age.

For each trait, circle the number that best indicates how accurately that trait describes you as you typically are. Choose from the following alternatives:

0=Not at All
Accurate

1=A Little
Accurate

2=Moderately
Accurate

3=Quite a Bit
Accurate

4=Extremely
Accurate

Untalkative	0	1	2	3	4
Anxious	0	1	2	3	4
Extraverted	0	1	2	3	4
Resentful	0	1	2	3	4
Bashful	0	1	2	3	4
Sad	0	1	2	3	4
Shy	0	1	2	3	4
Tense	0	1	2	3	4

Timid	0	1	2	3	4
Touchy	0	1	2	3	4
Talkative	0	1	2	3	4
Irritable	0	1	2	3	4
Introverted	0	1	2	3	4
Nervous	0	1	2	3	4
Quiet	0	1	2	3	4
Depressed	0	1	2	3	4

Appendix E

Rosenberg Self-Esteem Scale

Instructions: Below is a list of statements dealing with your general feelings about yourself. If you strongly agree, circle **SA**. If you agree with the statement, circle **A**. If you disagree, circle **D**. If you strongly disagree, circle **SD**.

1. On the whole, I am satisfied with myself.

SA A D SD

2. At times, I think I am no good at all.

SA A D SD

3. I feel that I have a number of good qualities.

SA A D SD

4. I am able to do things as well as most other people.

SA A D SD

5. I feel I do not have much to be proud of.

SA A D SD

6. I certainly feel useless at times.

SA A D SD

7. I feel that I'm a person of worth, at least on an equal plane with others.

SA A D SD

8. I wish I could have more respect for myself.

SA A D SD

9. All in all, I am inclined to feel that I am a failure.

SA A D SD

10. I take a positive attitude toward myself.

SA A D SD

Appendix F

CM-20

Instructions: Please answer the following questions about yourself by circling the “true” or “false”.

- T F 1. I think a great many people exaggerate their problems to get the sympathy and help of others.
- T F 2. I think most people would lie to get ahead.
- T F 3. Most people will use somewhat unfair means to gain profit or advantage, rather than to lose it.
- T F 4. It is safer to trust nobody.
- T F 5. Most people inwardly dislike putting themselves out to help other people.
- T F 6. People generally demand more respect for their own rights than they are willing to give for others.
- T F 7. People often disappoint me.
- T F 8. When someone does me wrong, I feel I should pay her/him back if I can, just for the principle of the thing.
- T F 9. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.
- T F 10. Some of my family have habits that bother and annoy me very much.
- T F 11. I can be friendly with people who do things which I consider wrong.
- T F 12. I don't blame people for trying to grab everything they can get in this world.
- T F 13. I do not blame a person for taking advantage of someone who lays herself/himself open to it.
- T F 14. I am not easily angered.
- T F 15. I would certainly enjoy beating a crook at her/his own game.
- T F 16. At times I have had to be rough with people who were rude or annoying.
- T F 17. There are certain people whom I dislike so much that I am pleased when they are in trouble for something they have done.
- T F 18. I am often inclined to go out of my way to win a point with someone who has opposed me.
- T F 19. I do not try to cover up my poor opinion or pity a person just so s/he won't know how I feel.
- T F 20. I strongly defend my own opinions.

Appendix G

Sleep (PSQI-abbrev.)

1. During the past month, what time have you usually laid down to go to sleep?

Bed time: _____ AM / PM

2. During the past month, what time have you usually gotten up in the morning?

Getting up time: _____ AM / PM

3. On an average night during the past month, how long has it usually taken you to fall asleep after you laid down to go to sleep?

Minutes to fall asleep: _____ minutes

4. On an average night during the past month, how many minutes of sleep did you lose because you woke up in the middle of the night?

Minutes of sleep lost at night: _____ minutes

5. On an average night during the past month, how many minutes of sleep did you lose because you woke earlier than your usual time to get up?

Minutes of sleep lost in morning: _____ minutes

6. During the past month, how would you rate your sleeping quality overall?

_____ (1) very bad _____ (2) fairly bad _____ (3) fairly good _____ (4)
very good

7. During the past month, how often have you had trouble staying awake while driving, eating meals, or engaging in social activity?

_____ (1) never
_____ (2) less than once a week
_____ (3) once or twice a week
_____ (4) three or more times per week

8. During the past month, how often have you taken naps during the day?

_____ (1) never
_____ (2) less than once a week
_____ (3) once or twice a week
_____ (4) three or more times per week

Appendix H

CHIPS-SF

Mark the number for each statement that best describes HOW MUCH THAT PROBLEM HAS BOTHERED OR DISTRESSED YOU DURING THAT PAST TWO WEEKS INCLUDING TODAY. Mark only one number for each item. At one extreme, **0 means that you have not been bothered by the problem.** At the other extreme, **4 means that the problem has been an extreme bother.**

HOW MUCH WERE YOU BOTHERED BY:

Aches and pains	0	1	2	3	4
Muscle tension	0	1	2	3	4
Cramps or stomach upset	0	1	2	3	4
Headache	0	1	2	3	4